```
L2        62168 FILE HCAPLUS
L3        20516 FILE BIOSIS
L4        16626 FILE EMBASE
L5          415 FILE WPIDS
L6          973 FILE JICST-EPLUS

TOTAL FOR ALL FILES
L7       156984 (ADENOVIR? E1A OR E1A PROTEIN OR ANTIGEN!(A) VIRAL(A)(TUMOUR OR
                TUMOR) OR ONCOGENE!(A) PROTEIN!(A) VIRAL OR TRANSCRIPT? FACTOR!
                OR ADENOVIRUS EARLY PROTEIN!)

=> dis his


     (FILE 'HOME' ENTERED AT 13:13:08 ON 31 MAY 2002)

     FILE 'HCAPLUS' ENTERED AT 13:13:58 ON 31 MAY 2002
               E PROTEINACEOUS/CT 5
               E ERYTHROPOIETIN/CT 5
               E E3+ALL/CT
               E POST TRANSLATIONAL/CT
               E E9+ALL/CT

     FILE 'MEDLINE, HCAPLUS, BIOSIS, EMBASE, WPIDS, JICST-EPLUS' ENTERED AT
     13:17:18 ON 31 MAY 2002
L1         56286 FILE MEDLINE
L2         62168 FILE HCAPLUS
L3         20516 FILE BIOSIS
L4         16626 FILE EMBASE
L5           415 FILE WPIDS
L6           973 FILE JICST-EPLUS
     TOTAL FOR ALL FILES
L7        156984 S (ADENOVIR? E1A OR E1A PROTEIN OR ANTIGEN!(A)VIRAL(A)(TUMOUR O

=> s l7 and (erythropoietin or glcyoprotein hormone or receptor!(a)erythropoietin?
or anemia disease or erythropoiesis)
L8           402 FILE MEDLINE
L9           684 FILE HCAPLUS
L10          159 FILE BIOSIS
L11          130 FILE EMBASE
L12           11 FILE WPIDS
L13            9 FILE JICST-EPLUS

TOTAL FOR ALL FILES
L14         1395 L7 AND (ERYTHROPOIETIN OR GLCYOPROTEIN HORMONE OR RECEPTOR!(A)
                 ERYTHROPOIETIN? OR ANEMIA DISEASE OR ERYTHROPOIESIS)

=> s l14 and (post translational or posttranslation? or peritranslational or peri
translation?)(1a)(modif? or process?)
L15            4 FILE MEDLINE
L16            4 FILE HCAPLUS
L17            0 FILE BIOSIS
L18            0 FILE EMBASE
L19            0 FILE WPIDS
L20            0 FILE JICST-EPLUS

TOTAL FOR ALL FILES
L21            8 L14 AND (POST TRANSLATIONAL OR POSTTRANSLATION? OR PERITRANSLATI
                 ONAL OR PERI TRANSLATION?)(1A)(MODIF? OR PROCESS?)

=> dup rem l21
PROCESSING COMPLETED FOR L21


Searched by: Mary Hale 308-4258 CM-1 12D16
```

L22 7 DUP REM L21 (1 DUPLICATE REMOVED)

=> d cbib abs 1-7;s l14 and protein and (post or peri)(w)translat? and (process? or modif?)

L22 ANSWER 1 OF 7 MEDLINE DUPLICATE 1
2001267722 Document Number: 21257718. PubMed ID: 11358837. Dynamic, site-specific interaction of hypoxia-inducible factor-1alpha with the von Hippel-Lindau tumor suppressor protein. Yu F; White S B; Zhao Q; Lee F S. (Department of Pathology, University of Pennsylvania School of Medicine, 605 Stellar-Chance Building, 422 Curie Boulevard, Philadelphia, PA 19104, USA.) CANCER RESEARCH, (2001 May 15) 61 (10) 4136-42. Journal code: 2984705R. ISSN: 0008-5472. Pub. country: United States. Language: English.
AB Hypoxia-inducible factor (HIF)-1alpha is a transcription factor that plays a critical role in regulating genes involved in **erythropoiesis** and angiogenesis. Recent evidence indicates that the von Hippel-Lindau tumor suppressor protein (VHL) is part of a ubiquitin ligase complex that promotes the degradation of HIF-1alpha under normoxic conditions. Under hypoxic conditions, HIF-1alpha is markedly stabilized. A critical issue in understanding the hypoxic response is the identification of hypoxia-regulated steps. We show here that hypoxia and cobalt treatment modulate the capacity of a HIF-1alpha fragment comprising residues 531-652 to coimmunoprecipitate with VHL. Hypoxia and cobalt both significantly diminish the interaction, and furthermore, normoxia treatment after hypoxia rapidly normalizes it. This HIF-1alpha fragment confers hypoxia and cobalt inducibility on a heterologous protein. Significantly, contained within this fragment is a short 27-residue sequence that behaves identically in all respects noted above. Finally, evidence is provided to show that cobalt and hypoxia both induce a **posttranslational modification** (or loss of one) in HIF-1alpha that affects its binding to VHL. We propose that dynamic, site-specific interaction of HIF-1alpha with VHL provides one mechanism by which HIF-1alpha can be regulated.

L22 ANSWER 2 OF 7 HCAPLUS COPYRIGHT 2002 ACS
2001:690745 Document No. 136:307331 Recent progress in Fanconi's anemia research: monoubiquitination of FANCD2 is the missing link between the Fanconi's anemia pathway and BRCA. Taniguchi, Toshiyasu (D'Andrea Laboratory, Department of Pediatric Oncology, Dana Farber Cancer Institute, Japan). Jikken Igaku, 19(14), 1901-1906 (Japanese) 2001. CODEN: JIIGEF. ISSN: 0288-5514. Publisher: Yodosha.
AB A review on candidate genes in Fanconi's anemia (FA) and a link between monoubiquitination of FANCD2 and BRCA1 in Fanconi's anemia. Topics discussed include characterization of Fanconi's anemia; candidate genes FANCA, FANCC, FANCD2, FANCE, FANCF, and FANCG; FA complex in Fanconi's anemia pathway; link between monoubiquitination of FANCD2 and BRCA1 in Fanconi's anemia; and biol. function of Fanconi's anemia pathway.

L22 ANSWER 3 OF 7 MEDLINE
2000243767 Document Number: 20243767. PubMed ID: 10758161. Hypoxia-inducible factor 1alpha protein expression is controlled by oxygen-regulated ubiquitination that is disrupted by deletions and missense mutations. Sutter C H; Laughner E; Semenza G L. (Institute of Genetic Medicine, Departments of Pediatrics and Medicine, The Johns Hopkins University School of Medicine, Baltimore, MD 21287-3914, USA.) PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (2000 Apr 25) 97 (9) 4748-53. Journal code: PV3; 7505876. ISSN: 0027-8424. Pub. country: United States. Language: English.
AB Hypoxia-inducible factor 1 (HIF-1) is a transcription factor that mediates cellular and systemic homeostatic responses to reduced O(2) availability in mammals, including angiogenesis, **erythropoiesis**, and glycolysis. HIF-1 activity is controlled by the O(2)-regulated expression

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of the HIF-1alpha subunit. Under nonhypoxic conditions, HIF-1alpha protein is subject to ubiquitination and proteasomal degradation. Here we report that missense mutations and/or deletions involving several different regions of HIF-1alpha result in constitutive expression and transcriptional activity in nonhypoxic cells. We demonstrate that hypoxia results in decreased ubiquitination of HIF-1alpha and that missense mutations increase HIF-1alpha expression under nonhypoxic conditions by blocking ubiquitination.

L22 ANSWER 4 OF 7 HCAPLUS COPYRIGHT 2002 ACS
1999:457101 Document No. 132:47942 Regulation of cellular 15-lipoxygenase activity on pretranslational, translational, and posttranslational levels. Kuhn, Hartmut; Heydeck, Dagmar; Brinckman, Roland; Trebus, Frank (Institute of Biochemistry, University Clinics Charite, Humboldt University, Berlin, D-10115, Germany). Lipids, 34(Suppl., Fatty Acids and Lipids from Cell Biology to Human Diseases), S273-S279 (English) 1999. CODEN: LPDSAP. ISSN: 0024-4201. Publisher: AOCS Press.
AB A review with 34 refs. with an emphasis on the author's results. In mammalian cells, enzymic lipid peroxidn. catalyzed by 12/15-lipoxygenases is regulated by pretranslational, translational, and **posttranslational processes**. In rabbits, rats, and mice induction of exptl. anemia leads to a systemic up-regulation of 12/15-lipoxygenases expression. In addn., interleukins-4 and -13 were identified as strong up-regulators of this enzyme in human and murine monocyte/macrophages and in the lung carcinoma cell line A549, and the interleukin-4(13) cell surface receptor as well as the signal transducer and activator of transcription 6 (STAT6) appears to be involved in the signal transduction cascade. On the level of translation, 15-lipoxygenase synthesis is blocked by the binding of regulatory proteins to a characteristic guanine-cytosine-rich repetitive element in the 3'-untranslated region of the rabbit 15-lipoxygenase mRNA, and the formation of such 15-lipoxygenase mRNA/protein complexes was identified as mol. reason for the translational inactivity of the 15-lipoxygenase mRNA in immature red blood cells. However, proteolytic breakdown of the regulatory proteins which were recently identified as hnRNP K and hnRNP E1 overcomes translational inhibition during later stages of reticulocyte maturation. For maximal intracellular activity, 12/15-lipoxygenases require a rise in cytosolic calcium concn. inducing a translocation of the enzyme from the cytosol to cellular membranes as well as small amts. of preformed hydroperoxides which act as essential activators of the enzymes. 12/15-Lipoxygenases undergo irreversible suicide inactivation during fatty acid oxygenation, and this process may be considered an element of down-regulation of enzyme activity. Suicide inactivation and proteolytic breakdown may contribute to the disappearance of functional 12/15-lipoxygenase at later stages of **erythropoiesis**.

L22 ANSWER 5 OF 7 HCAPLUS COPYRIGHT 2002 ACS
1998:704599 Document No. 130:33481 Proteasomes regulate **erythropoietin** receptor and signal transducer and activator of transcription 5 (STAT5) activation. Possible involvement of the ubiquitinated Cis protein. Verdier, Frederique; Chretien, Stany; Muller, Odile; Varlet, Paule; Yoshimura, Akihiko; Gisselbrecht, Sylvie; Lacombe, Catherine; Mayeux, Patrick (Institut Cochin de Genetique Moleculaire, INSERM U363, Universite Rene Descartes, Paris, F75014, Fr.). Journal of Biological Chemistry, 273(43), 28185-28190 (English) 1998. CODEN: JBCHA3. ISSN: 0021-9258. Publisher: American Society for Biochemistry and Molecular Biology.
AB Cis is an Src homol. 2 domain-contg. protein, which binds to the **erythropoietin** receptor and decreases **erythropoietin** -stimulated cell proliferation. We show that Cis assocs. with the second tyrosine residue of the intracellular domain of the **erythropoietin** receptor (Tyr401). Two forms of Cis with mol. masses of 32 and 37 kDa

were detected, and we demonstrate that the 37-kDa protein resulted from **post-translational modifications** of the 32-kDa form. Anti-ubiquitin antibodies recognized the 37-kDa form of Cis and the proteasome inhibitors N-acetyl-leucyl-leucyl-norleucinal and lactacystin inhibited its degrdn., showing that the 37-kDa form of Cis is a ubiquitinated protein, which seems to be rapidly degraded by the proteasome. In **erythropoietin**-stimulated UT-7 cells, the activation of the **erythropoietin** receptor and signal transducer and activator of transcription 5 (STAT5) was transient and returned to basal levels after 30-60 min of **erythropoietin** stimulation. In contrast, these proteins remained strongly phosphorylated, and STAT5 remained activated for at least 120 min in the presence of proteasome inhibitors. These expts. demonstrate that the proteasomes are involved in the down-regulation of the **erythropoietin** receptor activation signals. Because the proteasome inhibitors induced the accumulation of both the ubiquitinated form of Cis and the Cis-**erythropoietin** receptor complexes, our results suggest that the ubiquitinated form of Cis could be involved in the proteasome-mediated inactivation of the **erythropoietin** receptor.

L22 ANSWER 6 OF 7 MEDLINE

1999009652 Document Number: 99009652. PubMed ID: 9793257. Biology of **erythropoietin**. Lacombe C; Mayeux P. (Institut National de la Sante et de la Recherche Medicale, Unite 363, ICGM, Universite Rene Descartes, Paris, France.. lacombe@cochin.inserm.fr) . HAEMATOLOGICA, (1998 Aug) 83 (8) 724-32. Ref: 129. Journal code: FYB; 0417435. ISSN: 0390-6078. Pub. country: Italy. Language: English.

AB **Erythropoietin** (Epo) controls the proliferation, differentiation and survival of the erythroid progenitors. This cytokine was cloned in 1985 and rapidly became used for treatment of anemia of renal failure, opening the way to the first clinical trials of a hematopoietic growth factor. The clonage of one chain of the Epo receptor followed in 1989, thereby opening the research on intracellular signal transduction induced by Epo. Epo is synthesized mainly by the kidney and the liver and sequences required for tissue-specific expression have been localized in the Epo gene. A 3'enhancer is responsible for hypoxia-inducible Epo gene expression. HIF-1 alpha and beta proteins bind to this enhancer. Gene regulation by hypoxia is widespread in many cells and involves numerous genes in addition to the Epo gene. The Epo receptor belongs to the cytokine receptor family and includes a p66 chain which is dimerized upon Epo activation; two accessory proteins defined by cross-linking remain to be characterized. Epo binding induces the stimulation of Jak2 tyrosine kinase. Jak2 activation leads to the tyrosine phosphorylation of several proteins including the Epo receptor itself. As a result, different intracellular pathways are activated: Ras/MAP kinase, phosphatidylinositol 3-kinase and STAT **transcription factors**. However, the exact mechanisms by which the proliferation and/or the differentiation of erythroid cells are regulated after Epo stimulation are not known. Furthermore, target disruption of both Epo and Epo receptor showed that Epo was not involved in the commitment of the erythroid lineage and seemed to act mainly as a survival factor.

L22 ANSWER 7 OF 7 MEDLINE

96347539 Document Number: 96347539. PubMed ID: 8756628. A single tyrosine of the interleukin-9 (IL-9) receptor is required for STAT activation, antiapoptotic activity, and growth regulation by IL-9. Demoulin J B; Uyttenhove C; Van Roost E; DeLestre B; Donckers D; Van Snick J; Renauld J C. (Brussels Branch, Ludwig Institute for Cancer Research, Brussels, Belgium.) MOLECULAR AND CELLULAR BIOLOGY, (1996 Sep) 16 (9) 4710-6. Journal code: NGY; 8109087. ISSN: 0270-7306. Pub. country: United States. Language: English.

AB Interleukin-9 (IL-9), a T-cell-derived cytokine, interacts with a specific

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receptor associated with the IL-2 receptor gamma chain. In this report, we analyze the functional domains of the human IL-9 receptor transfected into mouse lymphoid cell lines. Three different functions were examined: growth stimulation in factor-dependent pro-B Ba/F3 cells, protection against dexamethasone-induced apoptosis, and Ly-6A2 induction in BW5147 lymphoma cells. The results indicated that a single tyrosine, at position 116 in the cytoplasmic domain, was required for all three activities. In addition, we observed that human IL-9 reduced the proliferation rate of transfected BW5147 cells, an effect also dependent on the same tyrosine. This amino acid was necessary for IL-9-mediated tyrosine phosphorylation of the receptor and for STAT activation but not for IRS-2/4PS activation or for JAK1 phosphorylation, which depended on a domain closer to the plasma membrane. We also showed that JAK1 was constitutively associated with the IL-9 receptor. Activated STAT complexes induced by IL-9 were found to contain STAT1, STAT3, and STAT5 **transcription factors**. Moreover, sequence homologies between human IL-9 receptor tyrosine 116 and tyrosines (of other receptors activating STAT3 and STAT5 were observed. Taken together, these data indicate that a single tyrosine of the IL-9 receptor, required for activation of three different STAT proteins, is necessary for distinct activities of this cytokine, including proliferative responses.

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L23           4 FILE MEDLINE
L24           4 FILE HCAPLUS
L25           1 FILE BIOSIS
L26           0 FILE EMBASE
L27           0 FILE WPIDS
L28           0 FILE JICST-EPLUS

TOTAL FOR ALL FILES
L29           9 L14 AND PROTEIN AND (POST OR PERI)(W) TRANSLAT? AND (PROCESS?
                OR MODIF?)

=> s l29 not l21
L30           1 FILE MEDLINE
L31           1 FILE HCAPLUS
L32           1 FILE BIOSIS
L33           0 FILE EMBASE
L34           0 FILE WPIDS
L35           0 FILE JICST-EPLUS

TOTAL FOR ALL FILES
L36           3 L29 NOT L21

=> dup rem l36
PROCESSING COMPLETED FOR L36
L37            1 DUP REM L36 (2 DUPLICATES REMOVED)

=> d cbib abs

L37  ANSWER 1 OF 1     MEDLINE                              DUPLICATE 1
1998179150  Document Number: 98179150.    PubMed ID: 9510527.  Oxygen sensing,
     hypoxia-inducible factor-1 and the regulation of mammalian gene
     expression. Ratcliffe P J; O'Rourke J F; Maxwell P H; Pugh C W.
     (Erythropoietin Group, Institute of Molecular Medicine, John Radcliffe
     Hospital, Headington, Oxford OX3 9DS, UK.. peter.ratcliffe@hammer.imm.ox.a
     c.uk) . JOURNAL OF EXPERIMENTAL BIOLOGY, (1998 Apr) 201 ( Pt 8) 1153-62.
     Ref: 67. Journal code: I2F; 0243705. ISSN: 0022-0949. Pub. country:
     ENGLAND: United Kingdom. Language: English.
AB   A great many aspects of the anatomy and physiology of large animals are
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constrained by the need to match oxygen supply to cellular metabolism and appear likely to involve the regulation of gene expression by oxygen. Some insight into possible underlying mechanisms has been provided by studies of **erythropoietin**, a haemopoietic growth factor which stimulates red cell production in response to hypoxia. Studies of hypoxia-inducible cis-acting sequences from the **erythropoietin** gene have led to the recognition of a widespread transcriptional response to hypoxia based on the activation of a DNA-binding complex termed hypoxia-inducible factor-1 (HIF-1). Perturbation of the transcriptional response by particular transition metal ions, iron chelators and certain redox-active agents have suggested a specific oxygen sensing mechanism, perhaps involving a haem **protein** in a flavoprotein/cytochrome system. In addition to **erythropoietin**, HIF-1-responsive genes include examples with functions in cellular energy metabolism, iron metabolism, catecholamine metabolism, vasomotor control and angiogenesis, suggesting an important role in the coordination of oxygen supply and cellular metabolism. In support of this, we have demonstrated an important role for HIF-1 in tumour angiogenesis. HIF-1 itself consists of a heterodimer of two basic-helix-loop-helix **proteins** of the PAS family, termed HIF-1alpha and HIF-1beta, although other closely related members of this family may also contribute to the response to hypoxia. We have fused domains of HIF-1 genes to heterologous **transcription factors** to assay for regulatory function. These experiments have defined several domains in HIF-1alpha which can independently confer the hypoxia-inducible property, and they suggest a mechanism of HIF-1 activation in which **post-translational** activation/derepression of HIF-1alpha is amplified by changes in HIF-1alpha abundance most probably arising from suppression of proteolytic breakdown. Pursuit of the mechanism(s) underlying these **processes** should ultimately lead to better definition of the oxygen-sensing **process**.

```
=> s hatteboer, g?/au,in;s verhulst, k?/au,in;s schouten, g?/au,in
'IN' IS NOT A VALID FIELD CODE
L38           0 FILE MEDLINE
L39           0 FILE HCAPLUS
L40           0 FILE BIOSIS
'IN' IS NOT A VALID FIELD CODE
L41           0 FILE EMBASE
L42           0 FILE WPIDS
L43           0 FILE JICST-EPLUS

TOTAL FOR ALL FILES
L44           0 HATTEBOER, G?/AU,IN


'IN' IS NOT A VALID FIELD CODE
L45           2 FILE MEDLINE
L46           8 FILE HCAPLUS
L47           2 FILE BIOSIS
'IN' IS NOT A VALID FIELD CODE
L48           2 FILE EMBASE
L49           1 FILE WPIDS
L50           0 FILE JICST-EPLUS

TOTAL FOR ALL FILES
L51          15 VERHULST, K?/AU,IN


'IN' IS NOT A VALID FIELD CODE
L52          13 FILE MEDLINE
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L53          23 FILE HCAPLUS
L54          16 FILE BIOSIS
'IN' IS NOT A VALID FIELD CODE
L55          14 FILE EMBASE
L56          21 FILE WPIDS
L57           0 FILE JICST-EPLUS

TOTAL FOR ALL FILES
L58          87 SCHOUTEN, G?/AU,IN

=> s l58 and l51
L59           0 FILE MEDLINE
L60           1 FILE HCAPLUS
L61           0 FILE BIOSIS
L62           0 FILE EMBASE
L63           1 FILE WPIDS
L64           0 FILE JICST-EPLUS

TOTAL FOR ALL FILES
L65           2 L58 AND L51

=> dup rem l65
PROCESSING COMPLETED FOR L65
L66           1 DUP REM L65 (1 DUPLICATE REMOVED)

=> d cbib abs

L66  ANSWER 1 OF 1  HCAPLUS  COPYRIGHT 2002 ACS        DUPLICATE 1
2000:756885   Document No. 133:318279  Manufacture of accurately processed
     proteins in human cell lines synthesizing adenovirus E1 and E2A tumor
     antigens.  Hateboer, Guus; Verhulst, Karina Cornelia;
     Schouten, Govert Johan; Uytdehaag, Alphonsus Gerardus Cornelis
     Maria; Bout, Abraham (Introgene B.V., Neth.).  PCT Int. Appl. WO
     2000063403 A2 20001026, 127 pp.  DESIGNATED STATES: W:  AE, AG, AL, AM,
     AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ,
     EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP,
     KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ,
     PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US,
     UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF,
     BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU,
     MC, ML, MR, NE, NL, PT, SE, SN, TD, TG.  (English).  CODEN: PIXXD2.
     APPLICATION: WO 2000-NL247 20000417.  PRIORITY: EP 1999-201176 19990415;
     EP 1999-204434 19991221.
AB   Methods of manufg. foreign proteins with complete and accurate
     post-translational processing in human cell lines are described.  Human
     cell lines have a .beta.-galactoside .alpha.2,6-sialyltransferase involved
     in sialylation that is absent from non-human mammalian cell lines.  Cells
     are immortalized by transformation with the E1 and E2A genes of human
     adenovirus, but without the integration of other genes of adenovirus.
     Such proteins may have advantageous properties in comparison with their
     counterparts produced in non-human systems like Chinese Hamster Ovary
     (CHO) cells.  The construction of a cell line carrying these antigen genes
     and the construction of an expression vector that used the cytomegalovirus
     immediate-early promoter and enhancer to express an erythropoietin gene is
     described.  The cell lines that can grow in suspension or attached to a
     substrate and the copy no. of the gene can be increased by amplification
     of the segment using methotrexate and a dihydrofolate reductase marker.
     The manuf. of normally sialylated, biol. active human erythropoietin is
     demonstrated.


=> log y
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COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	36.23	49.48

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-2.48	-2.48

STN INTERNATIONAL LOGOFF AT 13:28:12 ON 31 MAY 2002

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